Monday, March 19, 1990
4:00PM–5:30PM, Room 6
Myocardial and Pericardial Disease

ENDOMYOCARDIAL FIBROSIS (EMF). IS THE VENTRICULAR FIBROSIS EVOLUTIVE?

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According to some authors, EMF is an evolutive disease. To clarify this evolutive behaviour, we studied the cineven - triculography of 16 patients, 4 male and 12 female, aged between 11 and 60 years (mean 37.4 years). Each patient was submitted to at least 2 examinations, in an interval varying from 1 to 10 years (mean 4.6 years). Four patients were studied after surgery (resection of the ventricular fibrosis and replacement or plasty of an atrioventricular valve).

In none of the patients, increase or evolution of the ventricular fibrosis was noted, when the ventriculographies were compared. Furthermore, 3 patients underwent a necroscopic study in the late postoperative period, and again in none of them recurrence of the ventricular fibrosis was found.

These results suggest that there is not an evolutive character in EMF, confirming the hypothesis that the ventricular fibrosis in EMF is just the consequence of a previously healed disease,and not an evolutive process.

PERICARDIAL INFLUENCE ON CARDIAC GEOMETRY AND FILLING IN PATIENTS WITH SEVERE CHRONIC RIGHT VENTRICULAR PRESSURE OVERLOAD: AN INTRA-OPERATIVE STUDY USING TRANSESOPHAGEAL ECHOCARDIOGRAPHY

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Under conditions of acute RV pressure or volume overload the pericardium exerts a prominent effect on ventricular diastolic filling. However, the constraining effect of the pericardium in Pts with severe chronic RV pressure overload is unknown. We therefore studied its influence in 10 Pts undergoing surgery for chronic (symptom duration: 64±52 months, mean±SD) thromboembolic pulmonary hypertension (RV systolic pressure 86±19mmHg). Simultaneous intraoperative transesophageal echo Doppler and intracardiac pressure (P) of the RV and LV were obtained to compare end-diastolic (ED) dimensions, P and Doppler mitral inflow profiles before and after pericardiotomy (PER). No significant changes were noted in RV and LVEDP after PER (9±6 vs 11±6 and 7±6 vs 6±6mmHg). The LV ED area was unchanged after PER (short axis view, 2.3±0.7 vs 2.4±0.5cm^2; 4-chamber view, 13.1±3.8 vs 14.3±3.9cm^2) as was the RV ED area (4-chamber view, 23.7±4.9 vs 23.4±3.1cm^2). No significant changes after PER were seen in peak early (45.8±16.2 vs 44.6±17.7 cm/sec) or late (57.2±16 vs 55.6± cm/sec) Doppler mitral inflow velocities. These results demonstrate that the pericardium adapts well to chronic RV pressure overload and under these conditions has no significant influence on ventricular diastolic geometry or filling.

PERICARDIAL RESTRAINT AND DIASTOLIC FILLING IN THE NORMAL AND ABNORMAL LEFT VENTRICLE

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Although pericardial restraint has been demonstrated in normal LV function and LV dysfunction (LVD), its influence on diastolic filling (DF) has not been clearly delineated. We studied 6 closed chest post pericardiectomy (PECT) canines and 6 with intact pericardia (IP) at paced baseline and after induction of LVD with coronary microsphere embolization. Echo LV size and ejection fraction (EF), LV pressures, and transmitral Doppler were obtained at baseline, with LVD, and with volume loading (VL). At baseline, both groups had similar LV sizes, EF's, and LVEDP. The rapid filling fraction (RFF) was lower in the PECT group (62±8% vs 72±11%, p<.05). VL increased LV size 19% (p<.05) and LVEDP (6±3 vs 14±2 mmHg, p<.05) in the IP animals but did not alter DF. In the PECT group, VL increased LV size 22%, increased LVEDP (5±4 vs 12±3 mmHg), but decreased the RFF (62±8% vs 51±8%) and increased the atrial filling fraction (AFF) (28±6% vs 35±5%) (all p<.05). With LVD, both groups had similar LV sizes, EF's, and LVEDP. The RFF (54±9% vs 63±5%, p<.05) was lower and AFF (41±5 vs 36±3%, p<.05) higher in the PECT group. In the IP group, VL increased LV size 19%, LVEDP (13±4 vs 20±4 mmHg), RFF (63±5% vs 70±6%), but decreased AFF (36±3% vs 30±6%) (all p<.05) despite minimal mitral regurgitation (MR). In the PECT group, VL increased LV size 21% (p<.05) and LVEDP (14±2 vs 24±5 mmHg, p<.05) but did not alter the RFF or AFF despite moderate MR. Conclusion: In the normal and abnormal LV function, the pericardium restrains later DF resulting in increased early DF.

SUDDEN DEATH IN DILATED CARDIOMYOPATHY
Conventional versus Amiodarone Treatment

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Data from a prospective 3-year evaluation in 111 pts with idiopathic dilated cardiomyopathy (IDC, group A, 26-65 years) were compared to 30 consecutive pts (group B) who underwent an extended diagnostic workup (programmed electrical stimulation, PES, 48h-Holter, E, ECG, exercise testing) and which were randomized to amiodarone (200 mg/day) or no specific antiarrhythmic treatment. Pts were controlled every 3 months (mean follow-up: 24±4 months). In group A logistic regression analysis demonstrated as risk factors: frequent ventricular pairs and tachycardia (VP/VT), a low ejection fraction (EF) and atrial fibrillation; sudden death rate, SD: 17%/year. In group B 27/30 fullfilled one of these criteria; 19/30 pts had frequent ventricular arrhythmias (VA, >30 VA/h), 15/30 pts VP/VT. Incidence and severity of VA correlated with a low EF (<35%, p<0.01), but not with PES results. In group B 3/30 pts died from SD (all without amiodarone, p<0.05) and 2 pts died from heart failure. All pts who died, were best predicted by the presence of frequent VP/VT and an EF <35%, but not by the inducibility of VA during PES.

Risk stratification in pts with IDC is effective and should be used to guide antiarrhythmic therapy. Survival of pts with IDC improves after amiodarone.